Electronic Supplementary Material (ESI) for Lab on a Chip. This journal is © The Royal Society of Chemistry 2017

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Supplementary Figures

Fig. S1 Thrombosis-on-a-chip fabrication process. (a) The 3D model as seen directly after printing. (b) The blue support struts were removed with a scalpel; small "pinches" were sufficient to detach the struts due to their small diameter. PDMS was then cast into the mould-box (red dotted line). (c) PDMS was cured overnight at 60 °C, resulting in a 4mm chip thickness. After curing, the PDMS was separated from the mould box by cutting the box at the edges with a scalpel and breaking it open. The removal of the 3D printed model vessel was conducted in two steps using forceps, and without the need for bending the PDMS chip during removal. (d) First, the model was gently pulled at the outlet side, where it broke at the thinnest part of the stenosis or at the heel of the outlet in case of the healthy channels. (e) Afterwards, the rest of the 3D printed construct was removed from the inlet side. (f) The resulting full 3D PDMS microfluidic chip.

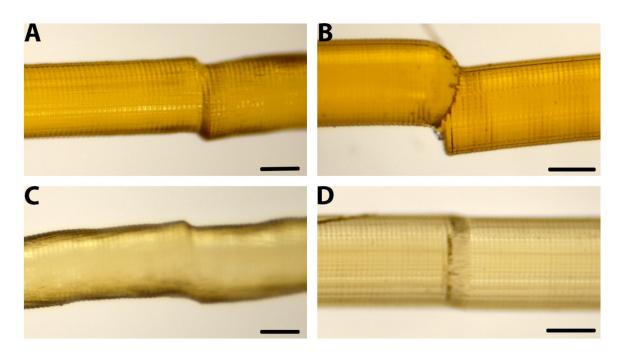


Fig. S2 Collection of printing defects presented in the PIC100 constructs. Scale bars, 200 μm

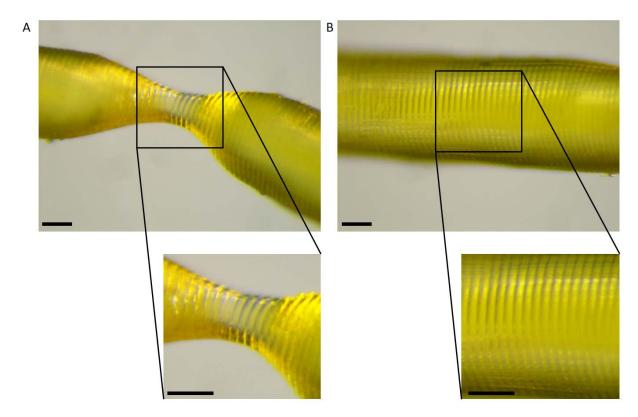


Fig. S3 Microscopy images of ring-like indentations produced in the SLA printing process. (a) Stenotic section. (b) Straight section. Scale bars, 100 µm.

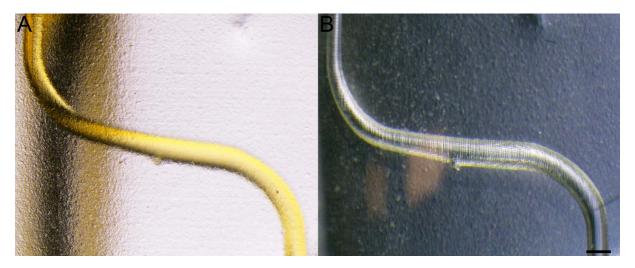


Fig. S4. Microscopy images of a 3D model incorporating two curves of approximately 80°, measured from the centre of curvature. (a) Close up of a print encapsulated in PDMS. (b) Close up of a cast PDMS chip after successful mould removal. Scale bar, 400 μm.

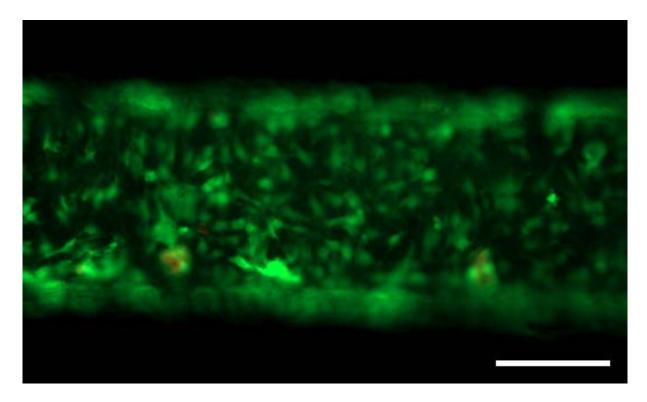


Fig. S5. Fluorescence microscopy images of a cytotoxicity measurement of HUVECs seeded in the 3D microfluidic chips. The HUVECs were stained using a live/dead staining. The number of dead cells is negligible. Scale bar, 200 μm.

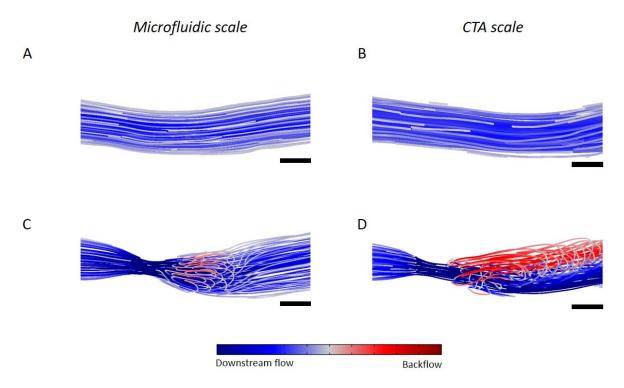


Fig. S6 Finite element analysis of streamlines simulated in healthy and stenotic vessels of 67% occlusion, simulated on microfluidic scale (a) (c) and CTA scale (b) (d). Healthy (a) (b) and stenotic (c) (d) vessel geometries were compared. An input flow of 1 ml/min and a vessel diameter of 4 mm were used for the CTA scale simulations and an input flow of 0.29 ml/min and a vessel diameter of 400 μ m for the microfluidic scale simulations. Both healthy geometries (a) (b) showed no sign of recirculation. The microfluidic scale simulation (c) showed minor recirculation while the CTA scale simulation showed a large recirculation area with elevated flow rates. Downstream flow directions are given in blue, stagnant flow is given in white and backflow is given in red. Scale bar (a) (c), 200 μ m. Scale bar (b) (d), 2 mm.

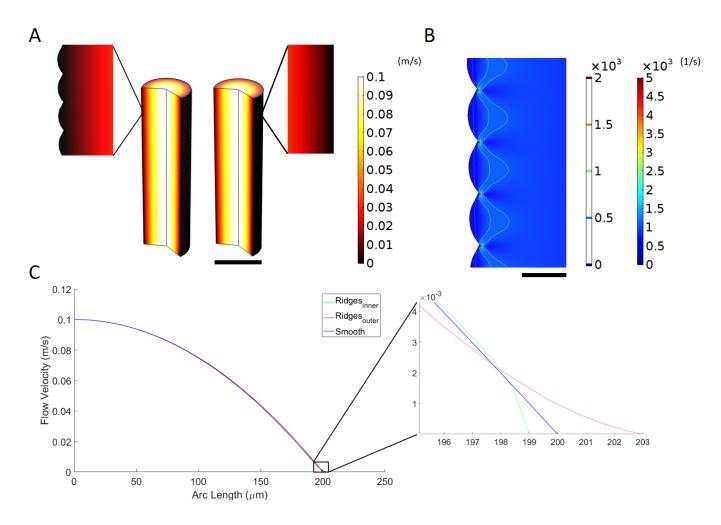


Fig. S7 Finite element analysis of the effect of ring-like indentations with a length of $25 \,\mu$ m (parallel to flow direction) and height of 4 μ m (perpendicular to flow direction). (a) Flow velocity profiles of a 1500 μ m section, with and without ring-like indentations respectively. Scale bar, 400 μ m. (b) Shear rate distribution of a channel with ring-like indentations showing a continuous scale and isolines with an equidistant spacing of 500 s⁻¹. A slightly elevated shear rate of 1500 s⁻¹ was measured on the inner part of the ridged structure. Scale bar, 25 μ m. (c) Flow velocity magnitude measured from the microfluidic channel centre to the outer surface. A comparison between the smooth geometry and the ridged geometry is given, where the velocity was measured up to either the inner part or the outer part of the ridge. Inset shows a magnified view of the outer 8 μ m of the channel showing almost equal flow velocities up to the outer 5 μ m.

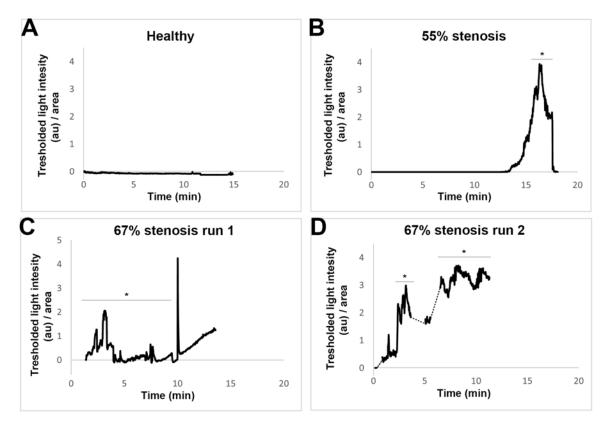


Fig. S8 Semi-quantitative platelet aggregation analysis. During perfusion the stenotic geometries revealed two aggregation mechanics, although each run depicted here revealed one predominant mechanism. (a) Healthy control revealing no platelet aggregation. (b) 55% stenotic geometry. Gradual aggregate growth around 14 minutes of perfusion and a steep drop after platelet dislodgement. Stick and slip mechanics were observed in the peak between the 15-18 minute time points (*). (c) 67% stenotic geometry. Mostly stick and slip growth, as shown by the multiple peaks and drops (*). Gradual growth of platelets was observed near the end of perfusion. (c) 67% stenotic geometry. A steep growth of platelets in the initial phase, followed by stick and slip growth (*). Missing data in (c-d, dotted lines) was intentionally left out because data was recorded out of focus or outside of region of interest. Stick and Slip regions are indicated with an asterisk (*).